

drugs. In each Figure, the CELx tests are shown to predict accurately whether a patient will or will not respond to a particular drug or combination of drugs except in one case. In FIG. 8B, it can be seen that one patient breast cancer cell sample that was expected to be a responder to gefitinib did not show a response in the CReMS testing.

**[0291]** The sensitivity and specificity of the CELx test for the patient cells and drug tested in Examples 1-4 as well as for the sub-groups of patients, drugs, pathways, and CReMS types tested is provided in FIG. 9. Overall and within each of the sub-groups studied, the CELx test generated high sensitivity (98%+) and specificity (99.9%+). These results illustrate the predictive power of the test across the different cancer cell types, drug types, CReMS types, and pathways targeted in the tests described in Examples 1-4.

What is claimed is:

1. A method of treating a human subject diagnosed with cancer, the method comprising:

administering to the subject a targeted therapeutic agent that targets a signaling pathway of interest in the subject's cancer cells, wherein the signaling pathway of interest has been determined to be active in the subject's cancer cells and sensitive to a targeted therapeutic agent that targets said pathway by a method comprising:

obtaining a sample of viable cancer cells from the subject; contacting a part of the sample with an activator agent of the signaling pathway of interest and determining whether there is a change in cell adhesion or attachment in the sample as compared to cell adhesion or attachment in the absence of the activator agent, wherein a change in cell adhesion or attachment indicates that the signaling pathway of interest is active in the subject's cancer cells; and

contacting a part of the sample with the activator agent and a targeted therapeutic agent that targets the signaling pathway of interest, and determining whether there is a change in cell adhesion or attachment in the sample as compared to cell adhesion or attachment in the presence of the activator agent alone, wherein a change in cell adhesion or attachment as compared to cell adhesion or attachment in the presence of the activator alone indicates that the signaling pathway of interest is sensitive to a targeted therapeutic agent that targets said pathway.

2. The method of claim 1, wherein the signaling pathway of interest is selected from the group consisting of MAPK-PK, RAS/RAF, RHO, FAK1, MEK/MAPK, MAK, MKK, AKT, EGF, HER2, HER3, HER4, PIK3/PTEN, VEGF, and combinations thereof.

3. The method of claim 1, wherein the activator agent is a ligand that binds to a cell surface receptor that modulates the signaling pathway of interest.

4. The method of claim 1, wherein the targeted therapeutic agent administered to the subject is selected from the group consisting of cetuximab, docetaxel, erlotinib, gefitinib, irinotecan, lapatinib, paclitaxel, pazopanib, topotecan, trastuzumab, fulvestrant, tamoxifen, letrozole, anastrozole, exemestane, everolimus, abiraterone, bicalutamide, bortezomib, vemurafenib, ipilimumab, and combinations thereof.

5. The method of claim 1, wherein the cancer is selected from the group consisting of breast cancer, lung cancer, and colon cancer.

6. The method of claim 1, wherein cell adhesion or attachment is measured using an impedance biosensor or an optical biosensor.

7. The method of claim 1, wherein the change in cell adhesion or attachment is assessed using Euclidean analysis.

8. The method of claim 7, wherein the Euclidean analysis is selected from the group consisting of arithmetic summation of the difference at multiple time points, temporal maxima, temporal minima, time to reach maxima or minima, changes in slope, absolute drop in biosensor signal, a total of all measurements, and combinations thereof.

9. The method of claim 1, wherein the change in cell adhesion or attachment is measured by a change in temporal maxima or minima.

10. The method of claim 1, wherein the change in cell adhesion or attachment is assessed using Euclidean analysis comprising arithmetic summation of the difference at multiple time points.

11. The method of claim 1, wherein the sample of viable cancer cells is cultured in a culture media free of serum prior to contact with the activator agent and/or the targeted therapeutic agent.

12. The method of claim 11, wherein the culture media comprises at least one growth factor.

13. The method of claim 11, wherein the culture media comprises at least one apoptotic agent.

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